THE DIABETIC FOOT

Table 58.1 Wagner's classification of diabetic foot lesions.

Grade 0	High risk foot with no ulceration or a pre-ulcerative lesion
Grade 1	Superficial ulcer (no deeper than skin) with no infection
Grade 2	Deeper ulcer penetrating to tendon or muscle, but no bone involvement. There may be cellulitis but no abscess formation
Grade 3	Deep ulcer with abscess or involvement (osteomyelitis)
Grade 4	Localised gangrene (toes, heel)
Grade 5	Extensive gangrene involving whole foot

Figure 58.1 Diabetic foot with flat base and prominent metatarsal heads. There is in-drawing of the toes ('hammer-toes'/ 'claw-toes') and chronically deformed joints at mid-foot and ankle (osterarthropathy) all leading to weight maldistribution and high propensity for injury and ulceration. There is a deep neuropathic ulcer on the heel.

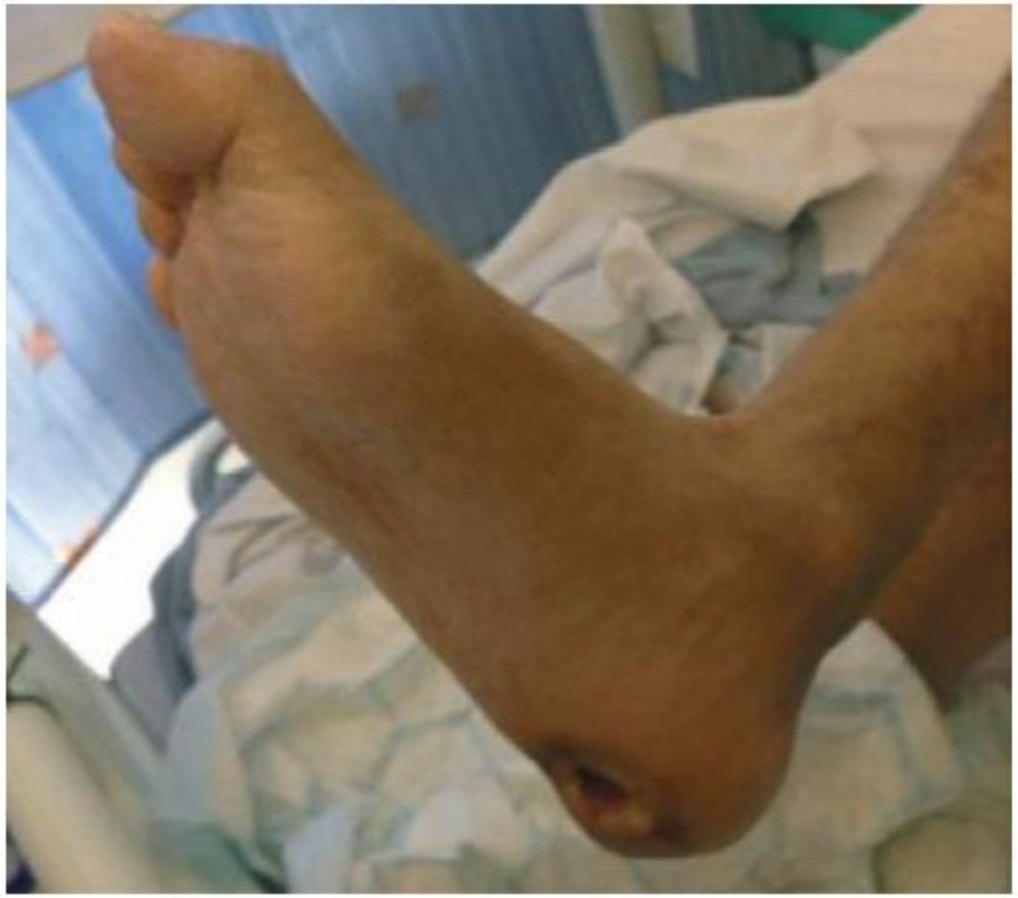


Figure 58.2 Diabetic dermopathy: dark brown non-blanching skin spots.

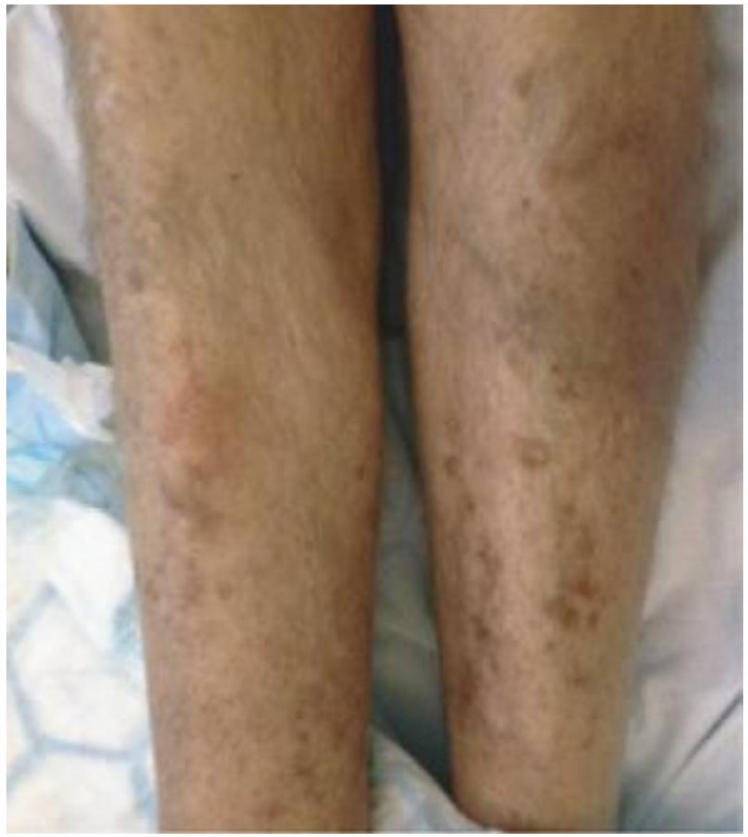


Figure 58.3 *Severe destruction secondary to infected diabetic foot. Wide debridement was performed followed by BKA.*

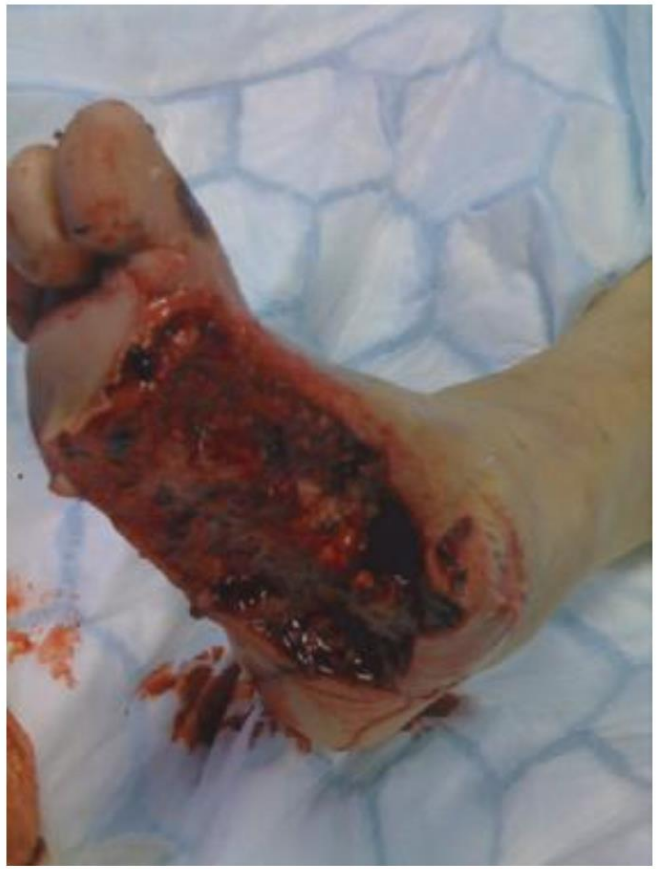


Figure 58.4 *Severe diabetic foot gangrene and infection. There has been wide debridement of the skin and tissue on the sole of the foot to manage acute infection with septicaemia.*

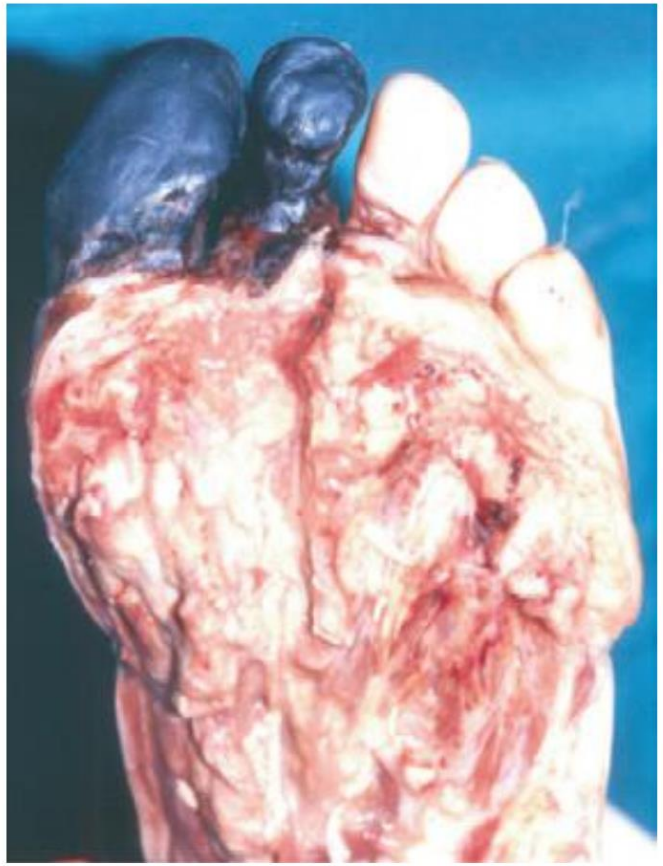


Figure 58.5 *Severe destruction and infection in a mixed ischaemia-neuropathic diabetic foot. Note the 'claw-like' toes and flat sole of foot. There has been an amputation of the 4th and 5th toe complex but with further 'die-back' of tissue from a combination of ischaemia and infection.*

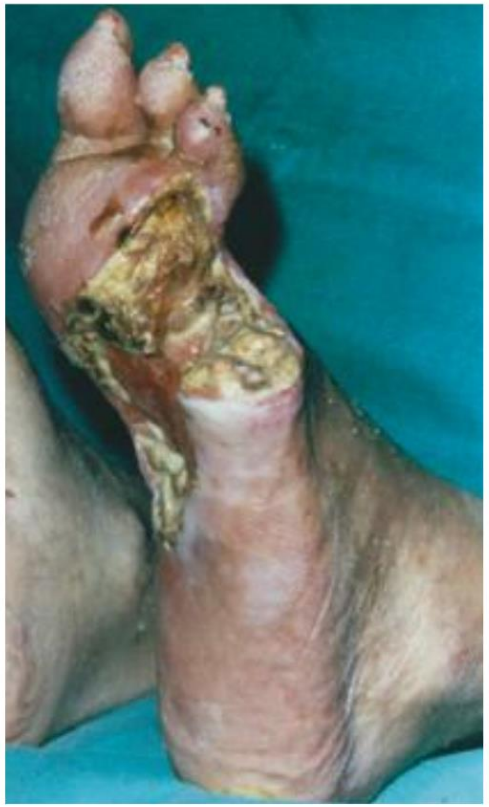
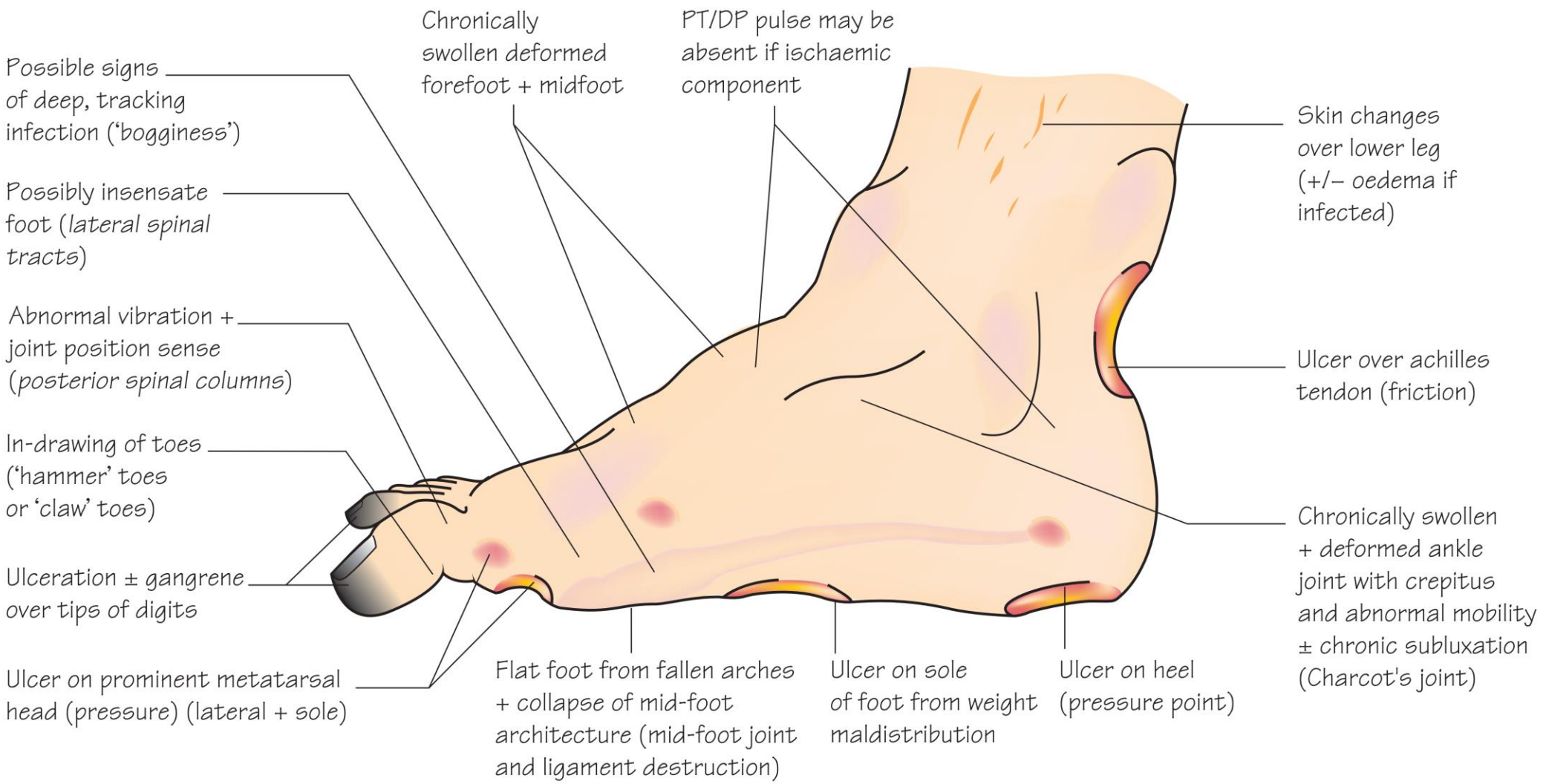


Figure 58.6 Clinical features of advanced diabetic foot.



Abbreviations: BKA, below knee; DP, dorsal pedis; PT, posterior tibial

- The diabetic foot represents a special problem for vascular surgeons and is truly a condition requiring a multidisciplinary team approach, including diabetology, ophthalmology, nephrology, podiatry, tissue viability and diabetic nurse specialists.

- **Aetiology**
  - Neuropathy.
  - Vasculopathy:
  - Macrovascular disease.
  - Microvascular disease.
  - Infection.

- **Neuropathy**

  Most common aetiology in both diabetic foot and diabetic ulceration.

  Pathophysiology. Diabetic neuropathy is complex. Often there is loss of the posterior spinal columns resulting in decreased joint position and vibration sense. Eventually there is a loss of the lateral spinal tracts leading to decreased sensation loss for pain and temperature, leaving the foot vulnerable to even trivial injury. In addition, microvascular disease affecting the peripheral nerves (including mononeuritis multiplex) leads to further loss of sensation and protective reflexes.

  Autonomic neuropathy, although leading to vasodilatation in the feet, also gives rise to microvascular shunting within cutaneous tissue, which is also thought to contribute to skin vulnerability, leading to ulceration and poor healing ability.

  Clinical features

  • Ulceration over pressure areas (heads of the metatarsal–phalangeal joints (especially first and fifth), lateral aspects of the sole and heel of the foot (often initiated by minor injury or chronic pressure/friction).

  • Chronic friction also leads to callus formation over pressure areas.

- **Vasculopathy**
  Pathophysiology. Both *macrovascular* and *microvascular* disease contribute to ischaemia in diabetic foot. Macrovascular disease refers to PVD, which is common in diabetes especially if patients are also smokers. The atherosclerosis is often diffuse and multilevel throughout the lower limb, particularly affecting the profunda (hence poorer ability to collateralise), and vessels are often smaller and heavily calcified (especially tibial vessels).
- Unreconstructable disease is more common compared with non-diabetic patients with PVD).
  Microvascular angiopathy is non-atherosclerotic disease affecting the vessels (skin, skeletal muscle, retina and kidney). It results in hyperglycaemia-related thickening of the basement membranes in the arterioles and capillaries leading to impaired oxygen diffusion. This microangiopathy contributes to the development of retinopathy, nephropathy and neuropathy seen in diabetes.

- **Clinical features**
  - Ischaemic ulceration and gangrene over friction and pressure points.
  - Infection is more common in diabetes, especially if there is a diabetic foot with ulceration. Infection and pus often track proximal through the foot, aggressively spreading to joint, bone and tendon if not promptly treated (open drainage and antibiotics).
  - Check the glycosylated HbA1c (long-term glucose control).
  - Diabetic nephropathy (monitor urea and creatnine).
  - Glycosuria and microalbuminuria (check with dipstick).
  - Diabetic retinopathy (regular ophthalmology review).
  - Skin pathology (ulceration [neuropathic/arterial]), diabetic dermopathy, necrobiosis lipodica diabeticorum, erythema nodosum)

- Examination of an Ulcer
  - An ulcer is a break of the continuity of an epithelium
  - Generally examination of an ulcer will follow the same pattern as examination of an lump (e.g. site, size, shape etc)

- **Base**- or floor of the ulcer may contain
  - Slough
  - Granulation tissue (capillaries, collagen, fibroblasts, inflammatory cells)
  - Deeper structures such as tendon or bone may also be visible
  - **Edge**- 5 types
  - Sloping (healing ulcer)
  - Punched out (commonly caused by diabetic neuropathy, peripheral arterial ischaemia)
  - Undermined (tuberculosis, pressure necrosis)
  - Rolled (basal cell carcinoma)
  - Everted (squamous cell carcinoma)
  - **Depth**-recorded by anatomically describing the structures it has penetrated
  - **Discharge**- may be serous, purulent. Always take a swab
  - **Relations**- may be adherent or invading deeper structure such as tendons, periosteum, bone.

  Local lymph glands may be enlarged (infection,malignancy)
  - **State of local tissue** (neurovascular examination

  especially important in lower limb ulcer